

REMARKS

I. Status of the Claims

Claims 1-22 and 24-26 have been canceled, and claim 23 is pending and stands rejected under 35 U.S.C. §112, first paragraph and §112 second paragraph. The specific grounds of rejection, and applicants' responses thereto, are set out in detail below.

II. Rejection Under 35 U.S.C. §112, Second Paragraph

According to the examiner, claim 23 is incomplete in reciting treatment without transgene expression. An amendment has been offered

III. Rejection Under 35 U.S.C. §112, First Paragraph

Claim 23 stands rejected as lacking an enabling disclosure in the specification. According to the examiner, the specification is defective in (a) failing to provide an adequate basis for predicting that an increase in α -MHC transcripts would benefit subjects having myocardial failure, (b) failing to provide correlation of α -MHC transgene expression *in vivo* with therapeutic benefit, and (c) failing to teach or provide guidance with respect to specific levels of α -MHC that would be therapeutic. Applicants provided an extensive response to which the examiner has, for the most part, simply "reiterated" the PTO's previous position. Thus, once again, applicants traverse.

First, applicants again submit that the examiner is incorrect in arguing that there is insufficient evidence that the increase in α -MHC transcripts seen in patients being successfully treated leads to patient benefit. When a messenger RNA level increases, it is common sense that

a commensurate increase in protein levels will follow. Of course, this is not always the case, but it is a rare occurrence when protein and message levels do not correlate. Thus, the burden should be on *the examiner* to explain why the demonstrated increase in message would not be viewed as predictive of therapeutic efficacy for α -MHC gene therapy, and thus supportive of enablement.

Moreover, applicants have supplemented the evidentiary record with a recent publication and a declaration on this very point.¹ The relied upon study examined MHC expression as a function of improved disease-state phenotype. This study showed a direct correlation between α - and β -MHC levels and a diseased heart state. Importantly, the age variation in the study subjects was minimal: 54.1 ± 10.5 years for tests and 49.1 ± 4.6 years for controls. Thus, the study was able to isolate the disease state as a variable and eliminate age as a complicating factor. The results, as attested to in the declaration, provide clear evidence that there is a direct, predictable and statistically relevant correlation between levels of α - and β -MHC and myocardial failure. Thus, absent compelling reason to doubt that the same benefit would be not achieved with gene therapy, enablement should be conceded.

Additionally, the examiner is referred to Jones *et al.*, *J. Clin. Invest.*, 98:1905-1917 (1996), which discusses the ablation of the α -MHC gene and the relationship between α -MHC protein and mRNA levels of α -MHC and LVEF, the measure of heart function applicants use as the standard for improvement in the currently prosecuted claim. Jones found that ablation of both α -MHC alleles led to gross heart defects that were partially rescued by the heterozygous form. This paper further showed that “mammalian left ventricular function can be severely compromised by a gene dosage effect involving” α -MHC, direct support of the notion that

increasing the levels of α -MHC (whether exogenously or endogenously) would be beneficial to LVEF. While this paper does not prove that exogenous addition of α -MHC can lead to increased LVEF, it strongly supports the inventor's paradigm, and as a result, coupled with later references regarding gene therapy in the heart, should be seen as sufficient to overcome the examiner's rejection regarding α -MHC.

Second, the examiner questioned what increase in α -MHC is needed to increase left ventricular ejection fraction, and thereby achieve therapy. The above referenced study used β -adrenergic blocking agents to improve the systolic function of subjects who exhibited the idiopathic dilated cardiomyopathy phenotype. The study measured expression levels of several genes and found a direct correlation with improvement in left ventricular ejection fraction and levels of α - and β -MHC. Specifically, as LVEF improved following treatment with β -adrenergic blocking agents, mRNA levels of α -MHC increased and mRNA levels of β -MHC levels decreased.

In a puzzling comment, the examiner states that "the Examiner's argument is not directed to the correlation of endogenous α -MHC expression with correlation to a disease phenotype." Apparently the examiner misapprehends applicants' previous statements. Applicants' point was that when one has (a) a baseline for normal α -MHC levels; (b) a disease state measure of α -MHC levels; and (c) monitored course of treatment levels for α -MHC, there is absolutely no basis for believing that one of skill in the art could not predict, with a reasonable expectation of success, what *in vivo* levels would be therapeutic. Thus, it is quite misleading and disingenuous to argue that "Applicant fails to show what levels of an α -MHC transgene expression are

¹ It is noteworthy that the §112 rejection in the companion '733 application was withdrawn in the face of this very evidence.

required to alleviate myocardial failure ...,” when such information is easily extracted from the information provided and the general knowledge in the field.

In conjunction with information provided in the application and the study described above, there is more than sufficient information on the baselines for normal and abnormal MHC expression. Moreover, the ability to track the elevation of α -MHC levels during the course of treatment provides a “real time” assessment of α -MHC levels as cardiac output improved. Thus, one can very readily determine at what point therapeutic efficacy is achieved. It also is not relevant that “complete amelioration” would be encompassed by therapy. The question is whether “therapy” is supported, and the numerous embodiments short of “complete amelioration” would be sufficient for this.

Moreover, the examiner’s comment that “Applicant has not provided guidance or evidence to *show* a correlation to therapeutic levels of expression of α -MHC transgene expression in an *in vivo* setting in a subject suffering from myocardial failure” shows that the examiner is effectively requiring clinical data. As the PTO has been repeatedly admonished by the CCPA and the Federal Circuit, it is *not* the FDA. It is the FDA’s province to approve therapeutic regimens. It is the PTO’s responsibility to determine whether a therapeutic method can be performed by one of skill in the art with a reasonable expectation of success. Here, there is overwhelming evidence that a decrease in α -MHC levels results in cardiac failure and, in the absence of some reason to expect that restoring these levels would not be therapeutic, enablement must be presumed.

Thus, ultimately, the rejection really appears to boil down to an argument over whether or not gene therapy for cardiac tissue is possible. Previously, applicants have provided a number of publications, far more relevant than those cited by the examiner, reporting on the successful

transfer of genes into cardiac tissue. Alexander *et al.*, *Clin. Exp. Pharmacol. Physiol.*, 26:661-668 (1999) reported gene transfer into myocardium through direct injection of plasmid DNA and viral transfer. Chien *et al.*, WO/2000/15821 describe the use of recombinant adenovirus-mediated expression of transgenes in both neonatal and mature cardiac tissues. Other papers reporting cardiac transgene expression included Davidson *et al.*, *Circulation* 104:131 (2001), Pachucki *et al.*, *Endocrinology* 142:13 (2001), Shinmura *et al.*, *Japan Heart J.* 41:633 (2000), Silva *et al.*, *FASEB* 14:1858 (2000), Lenhart *et al.*, *Am. J. Physiol. Heart Circ. Physiol.* 279:H986 (2000), Lazarous *et al.*, *Cardiovasc. Res.* 44:294 (1999), and Wickenden *et al.*, *Circ. Res.* 85:1067 (1999). Interestingly, other than to “reiterate” the previous argument, the examiner has failed to even mention this submission.

Despite the examiner’s failure to address these previous citations, applicants now provide yet additional references that undercut the examiner’s position on enablement. Fromes *et al.*, *Gene Therapy*, 12:683-688 (1999) describes the successful delivery of a gene to the myocardium by intraperitoneal injection. This paper starts by stating that “gene therapy is a potential new strategy for cardiovascular diseases” and goes on to state that “several studies have demonstrated the feasibility of gene transfer into the heart muscle.” In addition to this reference, a number of previous references demonstrated the potential of direct injection of genes into the myocardium (see Lin *et al.*, *Circulation*, 82:2217-2221 (1990); Stratford-Perricaudet *et al.*, *J. Clin. Invest.*, 90:626-630 (1992); Von Harsdorf *et al.*, *Circ. Res.*, 72:688-695 (1993); French *et al.*, *Circ. Res.*, 72:688-695 (1993); Lee *et al.*, *J. Thorac. Cardiovasc. Surg.*, 90:2414-2424 (1994); Coffin *et al.*, *Gene Therapy*, 3:560-566 (1996); and Kypson *et al.*, *J. Thorac. Surg.*, 115:623-630 (1998)). Nonetheless, Fromes constitutes an advance over these reports in developing a technique for “local delivery of the therapeutic gene into the pericardium,” demonstrating the successful

delivery of a gene to the heart. Fromes' results proved that "intra-pericardial injection ... leads to an efficient and safe strategy to deliver a transgene to the heart." The successful approach taken by Fromes came on the heels of another successful application of cardiovascular gene therapy by Hajjar *et al*, *Proc. Natl. Acad. Sci.*, 95:5251-5256 (1998). Hajjar used a catheter-based technique to successfully alter cardiac function in rat hearts. This study was seen as opening the prospect "of using somatic gene transfer to modulate overall cardiac function *in vivo*."

The success seen by Fromes and Hajjar has been built on by later researchers that validates the feasibility and effectiveness of cardiovascular gene therapy. Schroder *et al.*, *Transplantation*, 70:191-198 (2000) showed that addition of anti-CD4 monoclonal antibodies improved gene transfer into rat cardiac grafts. O'Donnell *et al.*, *Circ. Res.*, 88:415-421 (2001) showed that sarcoplasmic reticulum (SR) ATPase (SERCA), could be expressed in cardiac myocytes. del Monte *et al.*, *Circulation*, 104:1424-1429 (2001) also showed effective transfer of and expression of SERCA2a into a rat heart through adenoviral gene transfer. Li *et al.*, *Gene Ther.*, 21:1807-1813 (2003) showed that an adenoviral associated vector (AAV) could be successfully used to transfer a reporter gene and a therapeutic gene into the heart of a hamster. Lastly, applicants point to Yue *et al.*, *Circulation* (2003), who went yet a step further and actually treated a cardiovascular disease using an AAV vector to deliver a therapeutic gene to the heart of a diseased mouse. Yue not only delivered the gene but was able to see improvement of cardiovascular function and further saw improvement in disease state.

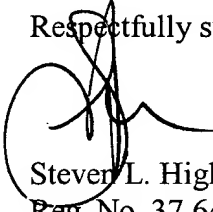
In short, applicants submit that the present record provides adequate evidence of the value of α -MHC therapy. In addition, the use of gene therapy in cardiac tissue is not so far

beyond the realm of possibility that it is non-enabled. Reconsideration and withdrawal is respectfully requested.

IV. Conclusion

In light of the foregoing, applicants respectfully submit that all claims are in condition for allowance, and an early notification to that effect is earnestly solicited. Should Examiner Ton have any questions regarding this response, a telephone call to the undersigned is invited.

December 23, 2003
Date

Respectfully submitted,

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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/558,472	04/25/2000	Michael R. Bristow	MYOG:004DIV1	8819

7590 12/04/2003

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EXAMINER

TON, THAIAN N

ART UNIT

PAPER NUMBER

1632

DATE MAILED: 12/04/2003

Please find below and/or attached an Office communication concerning this application or proceeding.

RECEIVED	
Date(s) Docketed:	<i>12/10/03 Response</i>
<i>Due, 12/10/03 email</i>	
<i>deadline</i>	
DEC 09 2003	
Client:	<i>MYOG:004DIV1</i>
Attorney(s):	<i>SLH / CRJ 12/04/03</i>
Initials:	<i>AM</i>

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UNDER SECRETARY OF COMMERCE FOR INTELLECTUAL PROPERTY AND
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Paper No.

Notice of Non-Compliant Amendment (37 CFR 1.121)

The amendment document filed on 10/02/03 is considered non-compliant because it has failed to meet the requirements of 37 CFR 1.121, as amended on June 30, 2003 (see 68 Fed. Reg. 38611, Jun. 30, 2003). In order for the amendment document to be compliant, correction of the following omission(s) or provision is required. Only the section (1.121(h)) of the amendment document containing the omission or non-compliant provision must be resubmitted (in its entirety), e.g., the entire "Amendments to the claims" section of applicant's amendment document must be re-submitted.

THE FOLLOWING CHECKED (X) ELEMENTS(S) CAUSE THE AMENDMENT DOCUMENT TO BE NON-COMPLIANT:

- ☐ 1. Amendments to the specification:
- ☐ A. Amended paragraph(s) do not include markings.
 - ☐ B. New paragraph(s) should not be underlined.
 - ☐ C. Other _____
- ☐ 2. Abstract:
- ☐ A. Not presented on a separate sheet, 37 CFR 1.72
 - ☐ B. Other _____
- ☐ 3. Amendments to the drawings: _____
- ☒ 4. Amendments to the claims:
- ☒ A. A complete listing of all of the claims is not present.
 - ☐ B. The listing of claims does not include the text of all claims (incl. withdrawn claims)
 - ☐ C. Each claim has not been provided with the proper status identifier, and as such, the individual status of each claim cannot be identified.
 - ☒ D. The claims of this amendment paper have not been presented in ascending numerical order.
 - ☐ E. Other: _____

For further explanation of the amendment format required by 37 CFR 1.121, see MPEP Sec. 714 and the USPTO website at <http://www.uspto.gov/web/offices/pac/dapp/opla/preognotice/officeflyer.pdf>.

If the non-compliant amendment is a **PRELIMINARY AMENDMENT**, applicant is given **ONE MONTH** from the mail date of this letter to supply the corrected section which complies with 37 CFR 1.121. Failure to comply with 37 CFR 1.121 will result in non-entry of the preliminary amendment and examination on the merits will commence without consideration of the proposed changes in the preliminary amendment(s). This notice is not an action under 35 U.S.C. 132, and this **ONE MONTH** time limit is **not extendable**.

If the non-compliant amendment is a reply to a **NON-FINAL OFFICE ACTION**, and since the amendment appears to be a *bona fide* attempt to be a reply (37 CFR 1.135(c)), applicant is given a **TIME PERIOD** of **ONE MONTH** from the mailing of this notice within which to re-submit the corrected section which complies with 37 CFR 1.121 in order to avoid abandonment. **EXTENSIONS OF THIS TIME PERIOD ARE AVAILABLE UNDER 37 CFR 1.136(a)**.

If the amendment is a reply to a **FINAL REJECTION**, this form may be an attachment to an Advisory Action. The period for response to a final rejection continues to run from the date set in the final rejection, and is not affected by the non-compliant status of the amendment.

W. Phillips
Legal Instruments Examiner (LIE)



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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/558,472	04/25/2000	Michael R. Bristow	MYOG:004DIVI	8819

7590 03/21/2003

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EXAMINER

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ART UNIT

PAPER NUMBER

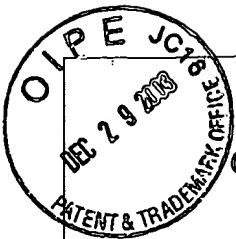
1632

DATE MAILED: 03/21/2003

Please find below and/or attached an Office communication concerning this application or proceeding.

RECEIVED
Date(s) Docketed: 6/24/03 Resp.
to OHA due: 9/12/03
Final Deadline
MAR 25 2003
Client: MYOG: 004US01
Attorney(s): SLH
Initials: [Signature] 352

10/27/03
4/2/03



Office Action Summary

Application No.

09/558,472

Applicant(s)

BRISTOW ET AL.

Examiner

Thai-An N. Ton

Art Unit

1632

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) ☒ Responsive to communication(s) filed on 13 January 2003.
- 2a) ☐ This action is FINAL. 2b) ☒ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

- 4) ☒ Claim(s) 23 is/are pending in the application.
- 4a) Of the above claim(s) _____ is/are withdrawn from consideration.
- 5) ☐ Claim(s) _____ is/are allowed.
- 6) ☒ Claim(s) 23 is/are rejected.
- 7) ☐ Claim(s) _____ is/are objected to.
- 8) ☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

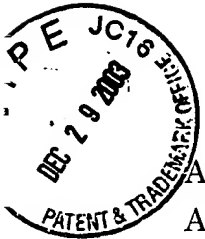
- 9) ☐ The specification is objected to by the Examiner.
- 10) ☒ The drawing(s) filed on 25 April 2000 is/are: a) ☒ accepted or b) ☐ objected to by the Examiner.
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
- 11) ☐ The proposed drawing correction filed on _____ is: a) ☐ approved b) ☐ disapproved by the Examiner.
If approved, corrected drawings are required in reply to this Office action.
- 12) ☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. §§ 119 and 120

- 13) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
a) ☐ All b) ☐ Some * c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
2. ☐ Certified copies of the priority documents have been received in Application No. _____.
3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).
* See the attached detailed Office action for a list of the certified copies not received.
- 14) ☒ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. § 119(e) (to a provisional application).
a) ☐ The translation of the foreign language provisional application has been received.
- 15) ☒ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. §§ 120 and/or 121.

Attachment(s)

- 1) ☐ Notice of References Cited (PTO-892) 4) ☐ Interview Summary (PTO-413) Paper No(s). _____
- 2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948) 5) ☐ Notice of Informal Patent Application (PTO-152)
- 3) ☐ Information Disclosure Statement(s) (PTO-1449) Paper No(s) _____ 6) ☐ Other:



Application/Control Number: 09/558,472

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DETAILED ACTION

Applicants' Amendment, filed 1/13/03, Paper No. 11, has been entered. Claim 23 has been amended.

Claim 23 is currently pending under examination.

Claim Rejections - 35 USC § 112

The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

The prior rejection of claim 23 under 35 U.S.C. 112, first paragraph, is maintained for reasons of record advanced on pages 2-6 of the prior Office action, mailed 11/27/01 (Paper No. 7).

The claim as amended is directed to a method of treating myocardial failure in a human comprising administering an effective amount of transgene encoding for α -MHC, wherein said treatment provides improvement in left ventricular ejection fraction.

The specification discloses a method of myocardial gene therapy to increase α -MHC expression by delivering a transgene encoding α -MHC to a human so that the α -MHC transgene is expressed in the myocardial tissue of the heart (see p. 14, lines 20-28 of the instant application). The specification further discusses construction of the transgene (p.15 of the specification) and modes of delivery of the transgene (p. 16, lines 4-15 of the specification). The specification specifically

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teaches up-regulation of α -MHC mRNA in myocardial tissue in human subjects suffering from cardiomyopathy, who received medical treatment with α -blocking agents (see example 5, of the instant application). It is reiterated that an increase in the amount of α -MHC mRNA in myocardial tissue does not provide a prediction of therapy for any subject having myocardial failure. Additionally, the specification fails to provide a correlation to therapeutic levels of expression of α -MHC transgenes in an *in vivo* setting in any subject having myocardial failure. Furthermore, the specification fails to teach or provide guidance for what level of α -MHC expression would provide a therapeutic effect in a human with myocardial failure, or how to measure the therapeutic effect in such a subject.

It is noted that the claim, as amended recites that the treatment provides an improvement in left ventricular ejection fraction. However, the specification fails to teach or show guidance for a correlation to therapeutic levels of expression of α -MHC transgenes *in vivo* such that improvement in the left ventricular ejection fraction would be improved. For reasons of record advanced in the prior Office actions, it is reiterated that the state of the art of gene therapy is unpredictable, and in particular, cardiovascular gene therapy is unpredictable. The specification fails to address how to overcome the unpredictabilities cited in the prior Office action, that are associated with the gene therapy art in general, and specifically as it pertains to the cardiovascular gene therapy. The rejection or question, in view of the guidance provided in the specification, is whether sufficient expression can be achieved by the exogenously administered α -MHC DNA sequence to have any

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effect of myocardial failure in a human, and in particular, the effect recited in the amended claim, that there would be an improvement in the left ventricular ejection fraction.

It is reiterated the Examiner's argument is directed to the unpredictable state of the gene therapy art, both in a general sense, and with particular regard to cardiovascular gene therapy, and furthermore, with particular regard to the expression of an α -MHC transgene; the Examiner's argument is not directed to the correlation of endogenous α -MHC expression with correlation to a disease-state phenotype. Furthermore, although Applicant provides an example of monitoring endogenous α -MHC mRNA levels *in vivo* to provide evidence to improved left ventricular ejection fraction, Applicant has not provided guidance or evidence to show a correlation to therapeutic levels of expression of α -MHC transgene expression in an *in vivo* setting in a subject suffering from myocardial failure; further, Applicant fails to show what levels of an α -MHC transgene expression are required to alleviate myocardial failure, or a protocol for reaching such levels

Thus it is maintained that the specification fails to enable the claimed invention for the reasons of record in the prior Office action (Paper No. 7) as discussed in the preceding paragraphs.

Claim Rejections - 35 USC § 112

The following is a quotation of the second paragraph of 35 U.S.C. 112:

The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

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The rejection of claim 23 under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention is maintained for reasons of record advanced on pp. 6-7 of the prior Office action (Paper No. 7).

Claim 23 is incomplete. It is further unclear how the step of the method, "administering an effective amount of a transgene encoding α -MHC," correlates to the intended effect of the method (the preamble), "treating myocardial failure" since, in light of specification, mere administration of an α -MHC transgene would not be sufficient to achieve treatment of myocardial failure without the expression of the recombinant DNA. Amendment to the claim is requested.

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Conclusion

No claim is allowed.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Thái-An N. Ton whose telephone number is (703) 305-1019. The examiner can normally be reached on Monday through Friday from 8:00 to 5:00 (Eastern Standard Time), with alternating Fridays off. Should the examiner be unavailable, inquiries should be directed to Deborah Reynolds, Supervisory Primary Examiner of Art Unit 1632, at (703) 305-4051. Any administrative or procedural questions should be directed to William Phillips, Patent Analyst, at (703) 305-3482. Papers related to this application may be submitted to Group 1600 by facsimile transmission. Papers should be faxed to Group 1600 via the PTO Fax Center located in Crystal Mall 1. The faxing of such papers must conform with the notice published in the Official Gazette, 1096 OG 30 (November 15, 1989). The CM1 Fax Center number is (703) 872-9306.

TNT

Thái-An N. Ton
Patent Examiner
Group 1632



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PRIMARY EXAMINER
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